Stranguria & Hematuria in a Mature Dog

Cynthia RL Webster, DVM, DACVIM (Small Animal)
Tufts University

Otto, a 6-year-old, 10-kg, castrated miniature schnauzer, was presented for stranguria and hematuria.

History

Otto was presented following 2 days of stranguria and hematuria. The owners noted no other clinical signs and stated that the dog always had a calm disposition and a healthy thirst. Routine vaccinations and parasitic preventive medications were up-to-date.

Examination

Otto was quiet but bright and alert. His BCS score was 5/9, although he was small for his breed. All physical examination parameters were within normal limits.

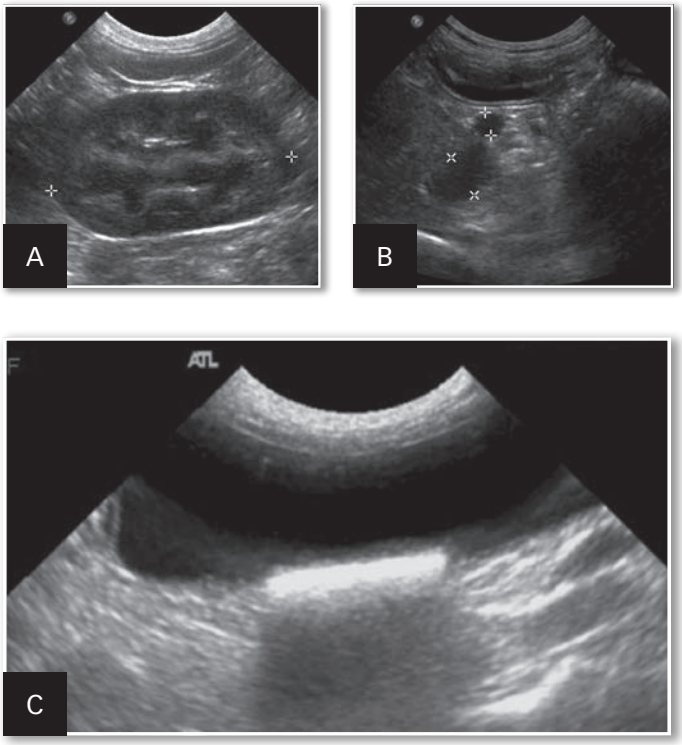
Diagnostics

Urinalysis revealed isosthenuria (urine specific gravity, 1.012), hematuria (RBCs too numerous to count), pyuria (10–20 WBCs per high-power field), bacteriuria (2+ rods), and ammonium biurate (1+) crystalluria. CBC was normal. Serum chemistry panel showed a mild increase in alanine transaminase (129 U/L; normal, <86 U/L). *Escherichia coli* was grown on urine culture.

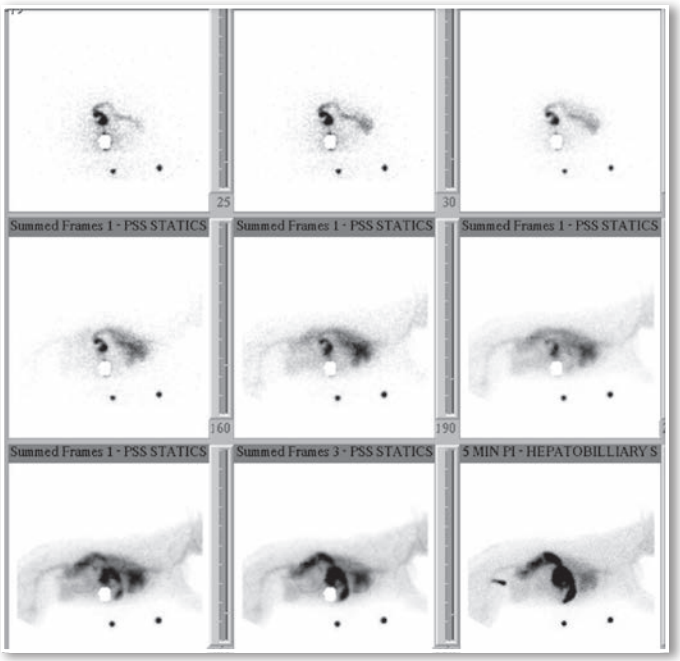
Abdominal radiographs were within normal limits. Abdominal ultrasonograpy was performed to further assess the urinary tract and liver and revealed an anomalous vessel leaving the portal vein and coursing dorsally and slightly caudally (**Figure 1**). Termination of the vessel could not be visualized, although a portoazygous (PA) shunt was suspected. The portal vein/caudal vena cava (PV/CVC) ratio was less than 0.54 (normal, >0.65). The PV/CVC ratio notes the luminal diameter of the portal vein to that of the aorta; with shunts, the portal vein becomes smaller, and thus the ratio decreases.

CPSS = congenital portosystemic shunt, HE = hepatic encephalopathy, PA = portoazygous, PV/CVC = portal vein/caudal vena cava

1 **Ultrasound changes suggestive of CPSS, including the presence of larger kidneys (ie, upper limit of normal; A), a PV/CVC ratio less than 0.54 (B), and bladder stones (C).**



2 **Transplenic portal scintigraphy using ultrasound guidance. With the dog under sedation, radioactive technetium pertechnetate (TCO) is injected into the spleen. Frames 1–9 (left to right) show the location of the TCO over time. In frame 1 most of the TCO is at the injection site in the spleen. By frames 2–4 the TCO is bypassing the liver and going directly to the heart via the shunting vessel. Only by frames 7–8 is TCO uptake in the liver visible.**



The liver was slightly small with decreased portal markings; the kidney/aorta ratio was 9.2 (range, 5.5–9.1), indicating that kidney size was at the upper limit of normal. Mineralized sediment noted in the bladder indicated the presence of multiple small uroliths.

Pre- and postprandial bile acids were 20 and 120 µM/L, respectively (range, <15 and 30 µM/L, respectively).

Diagnosis

Extrahepatic congenital portosystemic shunt (CPSS)

Extrahepatic CPSS was the suspected diagnosis because of the anomalous vessel on ultrasound, abnormal serum bile acids, and ammonium biurate crystalluria and ultrasonographic calculi. However, because the exact morphology of the probable CPSS could not be demonstrated on ultrasound, transplenic portal scintigraphy was pursued.[1] This study demonstrated the presence of portosystemic shunting (**Figure 2**).

Even in the absence of these scintigraphic findings, several lines of evidence virtually confirmed that this patient had a CPSS.

The miniature schnauzer is predisposed to CPSS,[2] and, compared with other predisposed breeds, the condition often presents later in life.[3,4] The combination of a small liver, large kidneys (ie, upper limit of normal), and uroliths on ultrasound has a positive predictive value of 100% for CPSS.[5] A PV/CVC ratio less than 0.65 and the presence of an anomalous vessel on ultrasound are also compatible with CPSS. Lastly, the increase in pre- and postprandial bile acids with no other signs of functional liver disease (eg, low albumin, high bilirubin) also is compatible with the presence of CPSS.

Ask Yourself

1. Does this dog need to be treated for hepatic encephalopathy (HE)?
2. Should Otto be treated medically or surgically?
3. Should this patient be started on anticonvulsant medication preoperatively?

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Treatment

Otto was started on a 2-week course of amoxicillin–clavulanic acid at 13.75 mg/kg q12h to treat the *E coli* urinary tract infection.

Initial medical management (**Table**) was continued for 4 weeks, after which the owners noted that the patient seemed more energetic. At surgery, the PA shunt was attenuated by placement of an ameroid constrictor. A cystotomy was performed at the same time, and multiple small (<3 mm) bladder stones were removed. A bladder wall culture and liver biopsy were performed. The dog was given preoperative levetiracetam.

Outcome

The perioperative period was uneventful. The bladder stones were 100% ammonium biurate in composition; bladder wall culture was negative. Liver biopsy showed typical histopathologic signs consistent with chronic hypoperfusion, including portal vein attenuation, duplication of hepatic arterioles, mild hepatic cord atrophy, mild lipidosis, and multiple lipogranulomas. Three weeks after surgery, the dog was clinically normal; postprandial total serum bile acids were also normal. Antiseizure and HE medications were tapered over the next month. Two years after surgery, Otto has had no urolithiasis.

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Did You Answer?

1. The presence of ammonium biurate crystals (and probably uroliths) indicates chronic hyperammonemia. The dog's compliant nature and polyuria/polydipsia may be manifestations of HE. The mild nature of the patient's clinical signs may have been partially caused by the PA shunt identified during surgery. Because PA shunts usually have a smaller diameter than portocaval shunts, often less blood bypasses the liver. Diaphragmatic movement during normal respiration in dogs with a PA shunt may intermittently compress and occlude the shunt.
2. The literature on medical management of CPSS was inadequate to help this decision. One older retrospective study suggested that older dogs with a normal blood urea nitrogen can do well medically.[6] A more recent prospective study showed that dogs treated surgically have longer survival times than those treated medically.[7] However, this study was not randomized and medical therapy was not standardized. The authors did suggest that some dogs with CPSS do well with medical management and concurred with preliminary results from a large retrospective study.[8] The factors that would allow us to predict whether an individual will manage well medically, however, have not been clearly delineated.

Medical management in this dog would involve treatment of HE, urolithiasis, and secondary bacterial urinary tract infection (**Table**). Without surgical removal, the dog's uroliths could result in urethral obstruction.

The ability of surgical attenuation of the CPSS (combined with cystotomy to remove uroliths) to cure the patient must be weighed against surgical risks. Mortality after attentuation of an extrahepatic CPSS by any method (eg, ameroid constrictor, ligation, cellophane banding) is similar, with a median mortality rate of 9.9% (range, 0%–27%).[9] Potentially fatal complications include postligation neurologic syndrome (PLNS), portal hypertension, sepsis, hemorrage, and thrombosis. Factors that predict poor outcome include hypoalbuminemia, leukocytosis, presence of HE at surgery, surgeon inexperience and, with ligation, the inability to completely attentuate the shunt at surgery. Early studies suggested that older age (>1 year) was a risk factor for mortality, but recent, more highly powered studies have not identified age as a risk factor.

Before surgery, it is important that the patient's HE be well controlled. Emerging evidence suggests that HE is a proinflammatory and potentially prothrombotic state in

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Table Preoperative Medical Management of CPSS

Intervention	*Dose*	*Possible Adverse Effects*	*Alternative(s)*
Diet (soy, dairy, vegetable sources, or protein; eg, Royal Canin Hepatic Support [royalcanin.us], Hill's Prescription Diet l/d Canine)	Start with 1.8–2.5 g/kg and titrate up	Too much protein can precipitate HE; too little can cause muscle catabolism and precipitate HE	Hill's Prescription Diet g/d Canine (hillsvet.com), Hill's Prescription Diet k/d Canine, Purina Veterinary Diets NF Kidney Function (purinaveterinarydiets.com)
Lactulose	0.5 mL/kg PO q12h titrated to achieve up to 3–4 soft stools a day	Diarrhea, dehydration	Lactitol at 250 mg/kg PO q12h
Levetiracetam	20 mg/kg PO q8h starting 2 days before surgery	Sedation	Phenobarbital at 3–5 mg/kg q12h for 3 weeks before surgery
Metronidazole	7.5 mg/kg PO q12h	Neurotoxicity (rare), granulocytopenia	Neomycin at 20 mg/kg PO q8–12h

dogs. It is possible that this may contribute to perioperative complications.[10,11] Treatment for HE (**Table**) is administered for a minimum of 3 to 4 weeks before surgery.

3. Older age may increase the occurrence of PLNS, which encompasses a constellation of neurologic signs ranging from twitches, tremors, and ataxia to life-threatening seizures. The incidence of PLNS after CPSS attentuation is around 6% (range, 0%–18%), with refractory seizures being less common (3.5%, range 0%–6.2%).[15] One study looking at surgical attenuation in dogs older than 5 years of age reported a 23% (4/17) incidence of PLNS, with 1 dog dying from refractory seizures and 3 with mild transient neurologic signs.[12] In another study in which 8 of 168 (4.7%) dogs had focal or grand mal seizures, 6 of the 8 were older than 1 year.[13] In yet another report, 4 of 5 dogs older than 18 months of age had postoperative seizures.[14] Thus this dog, by virtue of his older age, may benefit from preoperative antiseizure medication. A recent study suggests that initiation of levetiracetam (20 mg/kg PO q8h) for 1 or 2 days before surgery decreases the incidence of severe PLNS.[15] ■ cb

See **Aids & Resources**, back page, for references & suggested reading.